MYOCARDIAL CONTRACTILITY, AFTERLOAD MISMATCH AND VENTRICULAR DYSFUNCTION

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Although abnormalities in right ventricular function and/or the pulmonary vascular bed can be of pathophysiologic importance, it is more commonly an alteration of one or more determinants of left ventricular function which create an adverse hemodynamic status in the cardiac disease states encountered in man. Of the four major factors which control left ventricular function, i.e. heart rate, myocardial contractility, preload or end-diastolic fiber stretch, and afterload or the force which resists shortening, the latter is now appreciated as playing a particularly critical role in the mechanisms underlying heart failure. For example, the interdependence of wall shortening and afterload, at any given preload, has allowed understanding of the favorable influence of vasodilating agents in treating congestive heart failure. Moreover, the serial application of both invasive and non-invasive diagnostic techniques has provided improved insight into the role of afterload in chronic valvular heart disease, the potential for improving left ventricular shortening by valve surgery, and the relation between postoperative changes in afterload and regression of hypertrophy.

CONSTRUCTS FOR EVALUATION OF LEFT VENTRICULAR FUNCTION

There is a now a wealth of clinical studies which validate both angiographic (contrast or radionuclide) and echocardiographic methods for measurement of left ventricular volumes and dimensions, thereby providing relevant estimates of preload and myocardial shortening[1-3]. Quantitative assessment of afterload has been more problematic and described by one of two constructs (Table 1): 1) calculation of wall stress as a function of chamber dimension, pressure, and wall thickness[4-6] and 2) computation of aortic input impedance.
Table 1. Quantitative Descriptors of Left Ventricular Afterload

1. **Wall Stress**

\[ \sigma = \frac{P \times R}{2h} \]

Where \( P \) = intracavitary pressure, \( R \) = chamber radius, and \( h \) = wall thickness.

(The formula given applies to a spherical model)

2. **Aortic Input Impedance and Systemic Resistance**

- Impedance spectrum is the ratio of pressure (P) harmonics to corresponding flow (F) harmonics, derived from a Fourier analysis of the respective wave forms.

- Systemic resistance (R) equals the impedance modulus, or the ratio of amplitude of pressure to amplitude of flow, at the fundamental frequency (Heart rate) or 0 Hz. In a constant flow, non-pulsatile system, resistance can also be calculated as the ratio of mean pressure to mean flow.

derived from a Fourier analysis of ascending aortic pressure and flow[7-10]. With the latter approach, a spectral plot of the ratios of pressure to flow for each harmonic serves to describe comprehensively the input impedance moduli; however, it does not provide a single instantaneous or mean number which quantifies afterload. Thus, many investigators have chosen to use arterial resistance, calculated by dividing mean flow into mean pressure, or characteristic impedance obtained by averaging impedance moduli between 2 and 12 Hz[7,9,10]. The primary theoretical argument for use of aortic input impedance is that the physical properties of the systemic arterial system are constant during ejection and independent of cardiac function, which is contrary to the case with a wall stress analysis. While theoretically useful, measurement and computation of aortic input impedance, simultaneous with ventricular shortening, is technically difficult; moreover, a recent animal study from our institution would suggest that during aortic impedance changes, the corresponding alterations in wall stress and shortening characteristics are completely described within the framework of the traditional force-velocity-length relation[10]. Thus, in our clinical studies we have chosen to assess afterload using mean stress, i.e. the integral of the stress-time curve, during ventricular shortening.